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TI Interleukin 7 induces CD4+ T cell-dependent tumor rejection.
AU Hock H; Dorsch M; Diamantstein T; Blankenstein T
SO JOURNAL OF EXPERIMENTAL MEDICINE, (1991 Dec 1) 174 (6) 1291-8.

AB The potential of interleukin 7 (IL-7) to induce an antitumor response in vivo was analyzed. Therefore, the IL-7 gene was expressed in the plasmacytoma cell line J558L. Although the growth of IL-7-producing cells was not retarded in vitro, the IL-7-producing cells were completely rejected upon injection into mice. Tumor rejection was observed only in syngeneic but not in nude mice. The tumor-suppressive effect could be abolished by the parallel injection of an anti-IL-7 monoclonal antibody. Immunohistochemical analysis revealed IL-7-dependent infiltration of the tumor tissue by CD4+ and CD8+ T lymphocytes, and also type 3 complement receptor-positive (CR3+) cells, predominantly macrophages. Depletion of T cell subsets in tumor-bearing mice showed the absolute dependence of the antitumor response on CD4+ cells, whereas tumor rejection was unaffected by depletion of CD8+ cells. In addition to CD4+ cells, CR3+ cells were also needed for tumor rejection. The antitumor effect of IL-7 was confirmed by expression of the IL-7 gene in a second tumor cell line of different cellular origin. Together, our results demonstrate that a high local IL-7 concentration at the tumor site obtained by tumor cell-targeted gene transfer leads to tumor rejection involving a cellular mechanism that seems to be different from the ones observed in analogous experiments with other cytokines.

J558 used in vivo

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